August 2013 Case Study

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CC: Ankle pain

HPI: 16 year old previously healthy male presented with an ankle injury after landing awkwardly on his left ankle while playing basketball. He initially had mild lateral ankle pain, swelling and limping. While the swelling and limping decreased over the next few days, the pain became more noticeable over the anterior aspect of the ankle. The pain was dull and 0-1/10 at baseline, but became sharper, up to 6/10 with activity. The pain was exacerbated only by activity and relieved by rest and ibuprofen. He also noted that when he had his ankle taped prior to playing basketball, the pain essentially remained at its baseline level. When the pain did not completely resolve over the following week he was seen for further evaluation. He presented to the Primary Care Sports Medicine Clinic approximately two weeks after the initial injury. The remainder of the medical histories and review of systems were negative.

Physical Exam:
General: Afebrile, all vital signs within normal limits.
Gait: Nml
Left ankle:

  Inspection: Minimal swelling over left distal tibia
  Palpation: Tenderness overlying left distal tibia only.
  No tenderness over anterior talofibular, calcaneofibular, or deltoid ligaments.
  ROM: Full range of motion
  Strength: 5/5 with plantar flexion, dorsiflexion, inversion and eversion.
  Special tests: Negative anterior drawer test.
    Talar tilt negative & consistent with opposite side.
    Discomfort with squeeze test, but no tenderness over the anterior inferior
tibiofibular ligament.
    Kleiger’s test (external rotation/dorsiflexion of foot to stress deltoid ligament and
distal syndesmosis) negative.
    Hop test uncomfortable, but able to be performed

NV: Intact

Differential Diagnosis:
Lateral ankle sprain
Syndesmotic ankle sprain
Distal tibia fracture
Tibial stress fracture
Osteochondritis Dissecans (OCD) of talus
Tendinopathy (Tibialis anterior/posterior)
Imaging:

X-ray left ankle- Mild soft tissue swelling, no acute fracture. A 4.5 x 2.8 x 2.3 cm well defined circular distal tibial lesion with adjacent sclerosis seen.

Following the plain film the DDx was expanded to include the following:

- Unicameral bone cyst
- Osteoblastoma
- Osteosarcoma
- Aneurismal bone cyst
- Ewing’s sarcoma
- Osteoid osteoma
- Reticulosarcoma
- Multiple myeloma
- Giant cell tumor
- Acute osteomyelitis
- Brodie’s abscess (subacute osteomyelitis).
Images were reviewed with Orthopedic Surgery and there was concern that the lesion could represent more than a simple bone cyst. Because of the multi-loculated areas of lytic bone loss, along with the significant surrounding sclerosis, further characterization of the lesion was obtained with MRI.

MRI- left tibia with large lesion including metaphysis and physis, primarily intramedullary, approaching the cortex, with sclerotic margin present, and significant adjacent edema noted. It brightens considerably with T2-weighted signal.
The MRI was most consistent with a Brodie’s abscess based on location and character, so he was referred to Orthopedic Surgery for biopsy to confirm the diagnosis and exclude the abovementioned neoplasms. Labs were also drawn at that time.

**Labs:**
WBC 5.4, ESR 1, CRP 0.3.
Distal tibial biopsy culture: Positive for Staph aureus.

**Final/Working Diagnosis:** Brodie’s Abscess

**Treatment:** Incision and Drainage of tibial abscess. PICC line then placed for 4 weeks IV Clindamycin as an outpatient (based on culture sensitivities and infectious disease consultation).

**Outcome:** Postoperative course was uncomplicated. He was non-weight bearing immediately postoperatively for 4 weeks, but over the course of 10 weeks of physical therapy he returned to full, pain-free activity. He was followed clinically without issue, and following a subsequent ankle injury 18 months later his radiographs showed a well-healed tibia.

**Discussion:**

Brodie’s abscess is a form of subacute osteomyelitis first described by Sir Benjamin Brodie in 1832, characterized by its insidious clinical course. It is a relatively uncommon infection, with an incidence of approximately 2.9 out of 100,000. Persistent pain is the most common complaint, which may be present for weeks to months, often delaying its diagnosis.

As is typical of patients with Brodie’s abscess, our athlete was afebrile with normal inflammatory markers. Ezra et al described a case series of 16 patients of which only three had fever at time of presentation (18%), 2 had elevated CRP (12%), 5 had elevated ESR (31%), and 4 had leukocytosis (25%). Similarly, Harris and Kirkaldy-Willis found that in a study of nine patients, 3 had fever (33%), 4 had elevated ESR (44%), and all had a normal WBC. Though Brodie’s abscess can occur following direct trauma or surgical intervention, it usually occurs via hematogenous spread from a distant site. Its predilection for the metaphyses of long bones, most commonly the distal tibia, is due to the unique blood supply of growing bone. It is thought that fenestrations in the terminal ends of developing metaphyseal vessels allow for extravascular passage of bacteria. The surrounding bone tissue is relatively inaccessible to phagocytes which allows for bacterial proliferation. The most common offending organism is *Staphylococcus aureus*, found in 30-60% of positive cultures. Other common pathogens include *Staph epidermidis, Streptococcus, Pseudomonas, Kingella Kingae, H. influenza, and Pseudomonas*.

Diagnostic work up generally begins with plain radiographs. The lesion found in Brodie’s abscess classically has an area of central lucency surrounded by a sclerotic margin, which can be misdiagnosed in favor of a variety of neoplasms as listed above. Further imaging can be helpful to better characterize the lesion, but bone biopsy and subsequent histology showing inflammatory changes are necessary to make a definitive diagnosis. Even if an organism cannot be isolated, as is often the case, histology can differentiate an infectious from an oncologic process. Surgical curettage then is both diagnostic and therapeutic. Antibiotics are initiated following surgical debridement and after cultures are sent. Antimicrobial course is typically 4-6 weeks IV and based on susceptibilities if available.
Routine follow up imaging is often not necessary, as it often lags behind clinical resolution. However, some recommend that larger lesions be followed with serial radiographs.

References:


Brodie, BC. An account of some cases of chronic abscess of the tibia. Trans Med Chiro Soc. 17:238, 1832.


